

TCT-164**Persistent Endothelin Release After CABG But Not PCI: A Randomized Study Of Myocardial Injury After Revascularization for Complex Multivessel Disease**

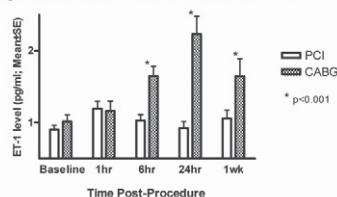
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Background: Endothelin (ET-1) is released after endothelial injury and is the most potent vasoconstrictor known in man. ET-1 is an adverse prognosticator in AMI, and has been shown to mediate ischemic myocardial injury. Both PCI and CABG induce ET-1 release however no study has compared ET-1 release after both procedures in a similar cohort. Additionally little is known about ET-1 release beyond the acute phase. This study aimed to compare patterns of ET-1 release after PCI or CABG, and correlate it with procedural injury.

Methods: Patients from a prospective randomized trial of PCI vs CABG were included. Procedural myocardial injury was assessed biochemically (troponin), and with gadolinium contrast MRI. Plasma ET-1 was measured at baseline, 1h, 6h, 24h and 1 week post-procedure.

Results: There were 37 PCI and 31 CABG patients. Baseline clinical characteristics were similar. A mean 4.2 ± 0.3 stents were implanted in the PCI group (total stent length 79 ± 5 mm). CABG was performed on-pump with an average of 1 mammary and 2 vein grafts. Both groups' ET-1 levels were similar at baseline and 1-hour. ET-1 levels post CABG then progressively increased and remain elevated at 1 week, whereas levels post PCI rapidly returned to baseline (Figure 1). ET-1 release as measured by area under curve was significantly greater for CABG compared to PCI (207 ± 23 v 378 ± 41 pg/ml-hr, p<0.001). ET-1 release did not correlate with troponin release (r=0.14, p=0.34), and was not significantly different in the 15 patients (6 PCI, 9 CABG) with MRI evidence of new infarction (319 ± 58 v 289 ± 30 pg/ml-hr, p=0.67).

Figure 1: Plasma ET-1 Post-PCI vs Post-CABG



Conclusion: ET-1 release patterns were clearly different after CABG compared to equivalent PCI in multivessel revascularization. ET-1 levels continue to rise up to 24 hours post-CABG and remain high at 1 week. ET-1 release did not correlate with biochemical or MRI evidence of new myocardial injury.

TCT-165**Incidence, Predictors, Management, Immediate and Long Term Outcomes Following Grade III Coronary Perforation**

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Background: Grade III coronary perforation is a rare but recognized complication of percutaneous coronary intervention (PCI).

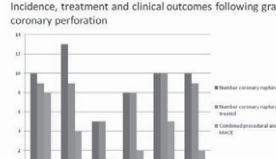
Methods: From a review of the procedural records of 24,465 patients, in 2 institutions from May 1993 to December 2009, 56 patients had PCI complicated by grade III coronary perforation.

Results: The majority of treated lesions were complex: 44.6% type B2, 51.8% type C and 28.6% chronic total occlusions; within the left anterior descending artery in 44.6 % and a small vessel (<2.5mm) in 32.1%. Glycoprotein IIb/IIIa inhibitors were administered in 17.9%.

The device causing perforation was an intracoronary balloon in 50%: 53.6% compliant, 46.4% non-compliant; intracoronary guide-wire in 17.9%, rotablation in 3.6%, and directional atherectomy in 3.6%. Following perforation, immediate treatment and success rates respectively were: prolonged balloon inflation 58.9%, 54.5%; covered stent implantation 46.4%, 84.6%; emergency coronary artery bypass surgery (CABG) 14.3%, 37.5%; and coil embolization 1.8%, 100%. Multiple methods were required to achieve hemostasis in 39.3%.

During the procedure (n=56), 19.6% required cardiopulmonary resuscitation and 3.6% died. In-hospital (n=54), 3.7% required CABG, 14.8% died. The combined procedural and in-hospital MI rate was 42.9%, and major adverse cardiac event (MACE) rate was 55.4%. At clinical follow-up (n=46) (median 38.1 months, range 7.6-122.8), 4.3% had an MI, 4.3% required CABG, and 15.2% died. At angiographic follow-up (56.5%), target lesion revascularization rate was 13%, target vessel revascularization rate 19.6%, and MACE rate 41.3% (Figure 1).

Figure 1: Incidence, treatment and clinical outcomes following grade III coronary perforation



Conclusions: PCI complicated by grade III coronary perforation is associated with complex lesions and high acute and long-term MACE rates.

TCT-166**Left Circumflex Occlusion in Myocardial Infarction: A review of the Catheterization and Percutaneous Coronary Intervention Registry (CathPCI Registry®)**

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Background: In contemporary practice, little is known about differences in outcomes between ST elevation myocardial infarction (STEMI) and non ST elevation myocardial infarction (NSTEMI) due to left circumflex or obtuse marginal (LCx/OM) occlusion. The incidence of ST elevation on ECG in MI secondary to LCx occlusion is 30-50%, potentially leading to worse outcomes.

Methods: We identified patients (pts) with STEMI or NSTEMI between 4/04 - 6/09 in the CathPCI Registry treated with PCI for culprit LCx/OM occlusion. Pts were included if they had 100% occlusion of LCx/OM, with the occlusion being the initial intervention during PCI. Prior coronary artery bypass grafting pts were excluded. Logistic generalized estimating equation modeling was used to compare in-hospital mortality between STEMI and NSTEMI pts, adjusting for baseline differences.

Results: Among 152,739 pts with LCx/OM occlusion, 127,591 (84%) presented with STEMI and 25,148 (16%) with NSTEMI. Baseline characteristics were similar between groups, despite statistically significant differences (Table). STEMI pts had larger infarcts, more cardiogenic shock, and higher adjusted mortality (OR 1.14, 95% CI 1.03-1.26, p=0.01) compared to NSTEMI pts.

Table: Comparison of STEMI vs. NSTEMI pts with LCx/OM occlusion

Patient Data	STEMI (n=127,591)	NSTEMI (n=25,148)	p-value
Admission			
Age	59 (51, 69)	58 (50, 68)	<0.0001
Symptom onset to admission (hrs)			NA
<6	107,629 (84.4%)	13,930 (55.4%)	
6-24	19,962 (15.6%)	11,218 (44.6%)	
Catheterization Lab			
Multivessel disease	64,979 (50.9%)	13,324 (53.0%)	<0.0001
Previously treated culprit	8,027 (6.3%)	1,444 (5.7%)	0.001
Post-procedure stenosis (%)			
<50	121,945 (95.6%)	23,203 (92.3%)	<0.0001
≥50	5,606 (4.4%)	1,941 (7.2%)	
# stents/lab visit			
0	13,011 (10.2%)	3,644 (14.5%)	<0.0001
1	72,696 (57.0%)	13,365 (53.2%)	
≥2	41,884 (32.8%)	8,139 (32.4%)	
Complete post-procedure TIMI flow	118,140 (92.6%)	22,655 (90.1%)	<0.0001
Ejection Fraction (%) ^a	45 (40, 55)	50 (40, 55)	<0.0001
Outcomes			
Death ^b	7,024 (5.8%)	728 (3.0%)	<0.0001
Cardiogenic Shock	3,210 (2.9%)	323 (1.4%)	<0.0001
Heart Failure	3,423 (3.0%)	350 (1.5%)	<0.0001
Peak CK-MB ^a	159 (70, 293)	89 (30, 200)	<0.0001

STEMI, ST elevation myocardial infarction; NSTEMI, non ST elevation myocardial infarction;

CK-MB, creatine kinase-MB

Continuous variables expressed as mean (IQR). % excludes missing data. Unless stated, missing data <1.0%

^a Missing up to 1/3 of data

^b Data excludes discharge status in 6,837 transfers

Conclusion: Total LCx/OM occlusion presents as NSTEMI less often than previously reported. NSTEMI pts with occlusion have better outcomes than STEMI pts, differing from prior studies indicating total occlusion of any coronary in the setting of NSTEMI confers equivalent risk to STEMI.

TCT-167**Risk Factors of Acute Radial Artery Occlusion Following Transradial Percutaneous Coronary Intervention in Senile Patients**

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Background: Compared with transfemoral percutaneous coronary intervention, the access site complication is less with transradial percutaneous coronary intervention (TRI). The acute radial artery occlusion (Acute RAO) is the most common complication following TRI. Although the incidence of acute RAO is less (0-10%) and acute RAO couldn't induce upper-extremity ischemia with positive Allen test patients, it makes re-TRI procedures impossible. To elucidate the risk factors of acute RAO is the best way to prevent it occurring.

Methods: A total of 1256 positive Allen test patients (≥60 years old) who underwent TRI (during May, 2004 to May, 2009) were divided into 2 groups: normal group and RAO group, according to whether the patient without or with acute RAO. Risk factors of acute RAO were analyzed by logistic regression model.

Results: Acute RAO occurred in 28 patients (2.2%). Univariate analysis showed, the larger size of sheath used, the higher incidence of acute RAO occurred. As compared with the patients in normal group, there are more female and diabetes mellitus patients in RAO group. The dose of heparin used in the operational procedure in RAO group were significantly less than normal group (3826±523IU vs 7425±980IU, p<0.01). The post-procedure duration of high-pressure compression hemostasis were longer in RAO group than normal group (378.9±35.4min. vs 264.7±43.2min., p=0.03). Multivariate logistic regression analyses showed that the dosage of heparin used in the procedure (odds ratio: 2.812, 95%CI: 1.116-6.732, p=0.016), size of sheath (odds ratio: 4.978, 95%CI: 3.211-10.675, p=0.001) and the post-procedure compression time (odds ratio: 2.431, 95% CI: 1.389-5.010, p=0.034) were all independent risk factors for acute RAO.

Conclusion: The incidence of acute RAO can be minimized by proper sheath selection, appropriate anticoagulation during operational procedure, and the avoidance of prolonged duration of high-pressure compression hemostasis following the procedure.